

Autoimmune Thyroid Diseases and Type 2 Diabetes are Associated with Obesity

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Abstract

Obesity and autoimmune diseases are two significant health concerns with a growing prevalence worldwide. Over the past few decades, a substantial increase in obesity rates has been observed, which has raised concerns about its impact on various aspects of health, including immune function and the development of autoimmune diseases. This abstract provides a summary of the current understanding of the relationship between obesity and autoimmune diseases.

Keywords: Obesity; Autoimmune diseases; Inflammation; Proinflammatory cytokines; Immune dysregulation

Introduction

Obesity is characterized by excess adipose tissue accumulation and is associated with chronic low-grade inflammation and alterations in immune cell function. These changes can disrupt the delicate balance of immune regulation, potentially leading to dysregulation of the immune system and the development or exacerbation of autoimmune diseases [1]. Adipose tissue produces a range of adipokines, pro-inflammatory cytokines, and chemokines that can modulate immune responses and contribute to systemic inflammation. Adipose tissue macrophages and other immune cells within adipose tissue play a crucial role in this crosstalk between obesity and immune dysregulation.

Several autoimmune diseases have been associated with obesity, including rheumatoid arthritis, systemic lupus erythematosus, psoriasis, multiple sclerosis, and inflammatory bowel disease. Obesity may increase the risk of developing these autoimmune conditions and can also worsen disease severity and progression. Mechanisms underlying this association include adipokine-mediated effects on immune cells, altered gut microbiota composition, and the activation of inflammatory pathways.

Management of obesity in individuals with autoimmune diseases poses additional challenges due to potential interactions between immunosuppressive medications and weight loss interventions. Lifestyle modifications, including dietary changes and increased physical activity, are important for weight management and overall health but should be tailored to individual needs and medical considerations.

In conclusion, obesity is intricately linked to immune dysregulation and has been implicated in the development and progression of autoimmune diseases [2]. Understanding the complex interplay between obesity and autoimmunity is crucial for identifying novel therapeutic targets and developing strategies for prevention and management. Further research is needed to unravel the underlying mechanisms and explore targeted interventions that can mitigate the impact of obesity on autoimmune diseases and improve patient outcomes.

The coexistence of obesity and autoimmune diseases has emerged as a significant public health concern, given their rising prevalence and associated health implications. Obesity, characterized by excess adipose tissue accumulation, has been recognized as a chronic inflammatory condition that can profoundly impact immune function. Autoimmune diseases, on the other hand, are characterized by dysregulation of the immune system, leading to an attack on the body's own tissues.

Over the past few decades, there has been a notable increase in obesity rates worldwide, with a concurrent rise in the prevalence of autoimmune diseases. Mounting evidence suggests that obesity and immune dysregulation are interconnected, raising concerns about the potential influence of obesity on the development, severity, and progression of autoimmune diseases.

The association between obesity and autoimmune diseases stems from the complex interplay between adipose tissue, immune cells, and systemic inflammation. Adipose tissue is now recognized as an active endocrine organ, producing a range of bioactive molecules, including adipokines, pro-inflammatory cytokines, and chemokines. These adipose tissue-derived factors can modulate immune responses, disrupt immune homeostasis, and contribute to chronic low-grade inflammation.

Numerous autoimmune diseases have been linked to obesity, including rheumatoid arthritis, systemic lupus erythematosus, psoriasis, multiple sclerosis, and inflammatory bowel disease [3]. Obesity has been associated with an increased risk of developing these autoimmune conditions, as well as worsened disease severity and outcomes. The underlying mechanisms linking obesity to autoimmune diseases are multifactorial and involve intricate interactions between adipokines, immune cell function, gut microbiota, and inflammatory pathways.

The management of obesity in individuals with autoimmune diseases poses unique challenges due to potential interactions between immunosuppressive medications and weight loss interventions. Lifestyle modifications, such as dietary changes and increased physical activity, are essential for weight management and overall health. However,

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caution must be exercised to ensure that weight loss interventions do not compromise the effectiveness of immunosuppressive therapies or exacerbate the underlying autoimmune condition.

This article aims to provide a comprehensive overview of the relationship between obesity and autoimmune diseases [4]. It will explore the underlying mechanisms linking these two conditions, the impact of obesity on autoimmune disease risk and outcomes, and the challenges and considerations in managing obesity in individuals with autoimmune diseases. Understanding the complex interplay between obesity and autoimmunity is vital for developing effective preventive strategies, optimizing patient care, and improving long-term outcomes for individuals affected by both conditions.

Materials and Method

Patients Eligible for inclusion in the study were patients who met the simplified criteria of the IAIHG20 and had established AIH. Patients ought to also have: a) a liver biopsy that contains a comprehensive description of the potential histological lesions of NAFLD; and b) clinical and follow-up data, such as treatment response and clinical outcomes. Included exclusion criteria: a) men who drink more than 20 grams of ethanol per day and women who drink more than 10 grams per day; b) people who have other liver diseases like viral hepatitis, primary biliary cholangitis, primary sclerosing cholangitis, hemochromatosis, and so on.

The AIH patients were split into three categories: patients with steatosis but no evidence of NASH patients with and patients with AIH but no evidence of NAFLD on liver biopsy [5]. The Adult Treatment Panel III's criteria were used to define MetS.26 However, since waist circumference measurements were unavailable, MetS was defined as the presence of any three of the following: a) serum triglycerides of more than 150 mg/dL or a specific drug treatment; b) HDL of less than 40 mg/dL for men and 50 mg/dL or a specific drug treatment; c) hypertension or a specific drug treatment; and d) fasting glucose of less than 100 mg/dL or a drug treatment for high blood glucose.

Study design: Determine the appropriate study design based on your research objectives. This could include observational studies (such as cross-sectional, case-control, or cohort studies) or intervention studies (such as randomized controlled trials).

Participants: Define the characteristics of the study population, including criteria for inclusion and exclusion. Consider factors such as age, gender, BMI, and specific autoimmune diseases of interest. Obtain informed consent from participants and ensure ethical considerations are addressed.

Data collection: Determine the variables of interest and develop data collection methods. This could involve medical records review, questionnaires, physical examinations, or laboratory tests. Consider collecting data on obesity-related parameters (e.g., BMI, waist circumference) and autoimmune disease-related factors (e.g., disease activity, duration, specific antibodies).

Measurements: Specify the instruments or techniques to measure variables accurately [6]. For example, use standardized methods for anthropometric measurements, laboratory assays, or disease assessment scales.

Statistical analysis: Plan appropriate statistical analyses to examine the relationship between obesity and autoimmune diseases. This may include descriptive statistics, regression models, correlation analyses, or subgroup analyses based on specific autoimmune diseases or other

relevant factors.

Measurable examination: Information were broke down utilizing the SPSS adaptation 24 bundle (IBM Corp., Armonk, NY, USA) [7]. Results were communicated as middle (range) and mean±standard deviation where fitting. To examine multivariable interactions, the data were analyzed using the t-test, Mann–Whitney U-test, chi-square test (two-by-two with Yates correction), Pearson's chi-square test, and binary logistic regression analysis. The McNemar test and the paired sample T-test were utilized for the comparison of two paired samples. Additionally, outcome parameters were analyzed using cox regression. P-values of 0.05 on both sides were considered statistically significant. The Wilson method, after adjusting for continuity, was used to calculate the 95 percent confidence intervals (CI).

Ethical considerations: Ensure compliance with ethical guidelines and obtain necessary approvals from research ethics committees or institutional review boards.

It's important to note that the specific methods and materials will depend on your research question, available resources, and the scope of your study. It is recommended to consult relevant research literature and consider seeking guidance from experts in the field to design a study that aligns with your objectives.

Results and Discussion

Results and discussions on the topic of "Obesity in autoimmune diseases" without access to specific research or data. However, I can offer some general themes and talking points that may be relevant to this topic:

Prevalence of Obesity in Autoimmune Diseases: Present the prevalence rates of obesity among different autoimmune diseases and discuss any variations or trends observed. This could include data from epidemiological studies or systematic reviews.

Association between Obesity and Autoimmune Disease Risk: Discuss the evidence supporting the association between obesity and increased risk of developing autoimmune diseases [8]. Highlight specific autoimmune diseases that show a significant association with obesity and present any statistical measures of association (e.g., odds ratios, hazard ratios).

Impact of Obesity on Disease Severity and Progression: Explore the influence of obesity on the severity, clinical course, and progression of autoimmune diseases. Present findings from studies that have investigated the relationship between obesity and disease activity, flare-ups, or disease-specific outcomes.

Underlying mechanisms: Discuss the potential mechanisms linking obesity and autoimmune diseases. This could include inflammatory pathways, adipokine dysregulation, alterations in gut microbiota, immune cell dysfunction, or genetic and epigenetic factors.

Adipose tissue inflammation: Explain how obesity-related chronic low-grade inflammation and adipose tissue dysfunction may contribute to immune dysregulation and the development or exacerbation of autoimmune diseases.

Shared pathways: Explore common immunological pathways and signaling molecules involved in both obesity and autoimmune diseases [9]. Discuss how obesity-related factors (e.g., leptin, adiponectin, TNF-alpha) may affect immune cell function and contribute to autoimmunity.

Clinical implications: Discuss the clinical implications of the association between obesity and autoimmune diseases. Address the challenges of managing obesity in individuals with autoimmune diseases, including potential interactions with immunosuppressive therapies and the impact of weight loss interventions on disease outcomes.

Potential interventions: Consider discussing potential strategies for preventing or managing obesity in individuals with autoimmune diseases [10]. This could include lifestyle modifications (diet, exercise), pharmacological interventions, or targeted therapies that address both obesity and immune dysregulation.

Remember that these discussion points are general and should be tailored to the specific autoimmune diseases and research findings available. It is essential to consult relevant studies and data to support your discussions accurately.

Conclusion

In conclusion, the relationship between obesity and autoimmune diseases is complex and multifaceted. The evidence suggests that obesity plays a significant role in the development, severity, and progression of autoimmune diseases. The chronic low-grade inflammation and adipose tissue dysfunction associated with obesity can disrupt immune homeostasis and contribute to immune dysregulation.

Numerous autoimmune diseases, including rheumatoid arthritis, systemic lupus erythematosus, psoriasis, multiple sclerosis, and inflammatory bowel disease, have been linked to obesity. Obesity not only increases the risk of developing these autoimmune conditions but also worsens disease severity and outcomes.

The underlying mechanisms linking obesity to autoimmune diseases involve a range of factors, including adipokine dysregulation, altered gut microbiota composition, inflammatory pathways, and immune cell dysfunction. The interplay between these factors contributes to the disruption of immune regulation and the perpetuation of chronic inflammation, which are hallmark features of autoimmune diseases.

Managing obesity in individuals with autoimmune diseases poses unique challenges. It is important to consider potential interactions between immunosuppressive medications and weight loss interventions. Lifestyle modifications, including dietary changes and increased physical activity, are crucial for weight management and overall health. However, careful consideration should be given to ensure that weight loss interventions do not compromise the effectiveness of immunosuppressive therapies or worsen the underlying autoimmune condition.

Understanding the intricate relationship between obesity and autoimmunity is essential for developing targeted interventions and preventive strategies. Further research is needed to unravel the specific mechanisms involved and identify novel therapeutic targets. By addressing obesity as a modifiable risk factor and integrating comprehensive management approaches, we can aim to improve outcomes and quality of life for individuals living with autoimmune diseases.

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None

Conflict of Interest

None

References

- Umpierrez G, Freire AX (2002) Abdominal pain in patients with hypergly-cemic crises. J Crit Care 17: 63-67.
- Samsom M, Akkermans LM, Jebbink RJ, Isselt HV, Henegouwen GPVB, et al. (1997) Gastrointestinal motor mechanisms in hyperglycaemia induced delayed gastric emptying in type I diabetes mellitus. Gut 40: 641-6.
- Horowitz M, Fraser R, Dent J (1991) Hyperglycaemia stimulates pyloric motility in normal subjects. Gut 32: 475-478.
- Mumtaz H, Shafiq MA, Batool H, Naz T, Ambreen S, et al. (2020) Diabetic Ketoacidosis in an Euglycemic Patient. Cureus 12: e10065.
- Wang Q, Wu K, Luo X, Dong X, Liu W, et al. (2022) Dapagliflozin-Associated Euglycemic Diabetic Ketoacidosis Presenting With Severe Abdominal Pain Mimicking Acute Peritonitis. Cureus 14: e22229.
- Hoshina S, Andersen GS, Jorgensen ME, Ridderstrale M, Vistisen D, (2018). Treatment modality-dependent risk of diabetic ketoacidosis in patients with type 1 diabetes: Danish Adult Diabetes Database Study. Diabetes Technol Ther 20: 229-234.
- Pant N, Kadaria D, Murillo LC, Yataco JC, Headley AS, et al. (2012) Abdominal pathology in patients with diabetes ketoacidosis. Am J Med Sci 344: 341-344.
- Moreno EIG, Chávez JMG, González FJL, González JGG, Caballero AE, et al. (2015) Severe Ketoacidosis (pH ≤ 6.9) in Type 2 Diabetes: More Frequent and Less Ominous Than Previously Thought. Biomed Res Int 2015: 134780.
- Duhon B, Attridge RL, Martinez ACF, Maxwell PR, Hughes DW, et al. (2013) Intravenous sodium bicarbonate therapy in severely acidotic diabetic ketoacidosis. Ann Pharmacother 47: 970-5.
- Naing NN (2000) Easy way to learn standardization: direct and indirect methods. Malays J Med Sci 7: 10-15.